# Postural Response to Vibration of Triceps Surae, but Not Quadriceps Muscles, Differs between People with and without Knee Osteoarthritis

Camille J. Shanahan,[1,2] Tim V. Wrigley,[1] Michael J. Farrell,[2] Kim L. Bennell,[1] Paul W. Hodges[3]

[1]Department of Physiotherapy, University of Melbourne, Melbourne, Victoria, Australia, [2]The Florey Institute of Neuroscience and Mental Health, Melbourne, Victoria, Australia, [3]Centre for Clinical Research Excellence in Spinal Pain, Injury and Health, School of Health and Rehabilitation Sciences, The University of Queensland, Brisbane, Queensland 4072, Australia

*Received 28 November 2013; accepted 2 April 2014*
*Published online 5 May 2014 in Wiley Online Library (wileyonlinelibrary.com). DOI 10.1002/jor.22637*

**ABSTRACT:** Although proprioceptive impairments are reported in knee osteoarthritis (OA), there has been little investigation of the underlying causes. Muscle spindles make an important contribution to proprioception. This study investigated whether function of quadriceps, triceps surae, and tibialis anterior muscle spindles is altered in individuals with knee OA. Thirty individuals with knee OA (17 females, $66 \pm 7$ [mean $\pm$ SD] years) and 30 healthy asymptomatic controls (17 females, $65 \pm 8$ years) stood comfortably and blindfolded on a force plate. Mechanical vibration (60 Hz) was applied bilaterally over the quadriceps, triceps surae, or tibialis anterior muscles for the middle 15 s (Vibration) of a 45 s trial (preceded and followed by 15 s Baseline and Recovery periods). Two trials were recorded for each muscle site. Mean anterior–posterior displacement of centre of pressure was analysed. Although there were no differences between groups for trials with vibration applied to the quandriceps or tibialis anterior, participants with knee OA were initially perturbed more by triceps surae vibration and accommodated less to repeated exposure than controls. This indicates that people with knee OA have less potential to detect or compensate for disturbed input to triceps surae, possibly due to an inability to compensate using muscles spindles in the quadriceps muscle. © 2014 Orthopaedic Research Society. Published by Wiley Periodicals, Inc. J Orthop Res 32:989–996, 2014.

**Keywords:** knee; osteoarthritis; proprioception; muscle spindles; postural control

Knee osteoarthritis (OA) is a common musculoskeletal condition characterized by pain, physical disability, altered movement and sensation, and psychological distress.[1] Although changes in movement and muscle activation in knee OA are likely to be multifactorial, recent work provides evidence of an association between proprioceptive impairment and both pain and functional limitations.[2–7] Many studies[8–15] indicate impaired proprioceptive acuity of both affected and unaffected knees and other joints in the presence of knee OA. Despite the potential importance of impaired proprioception for accurate control of joint motion and stability, there has been little investigation of the underlying mechanisms of proprioceptive dysfunction. Better understanding of the causes of proprioceptive impairments associated with knee OA may inform optimized treatments for this condition.

Although altered afferent input from mechanoreceptors in the joint (the site of primary pathology in OA) may seem the most likely candidate to explain compromised proprioception, muscle spindle function may also be compromised. Muscle spindles provide a critical contribution to perception of joint position and movement.[16–20] Changes in muscle spindle function in knee OA could be mediated by a range of sequelae of the disease including structural changes to the muscles in which they are situated (in parallel with muscle fibres), changes to the intrafusal muscle fibres (muscle fibres within the muscle spindle), or effects on the sensory elements of the spindle. Knee OA involves atrophy and morphologic changes of the quadriceps muscle. Quadriceps muscle cross sectional area is ~12% smaller on the side of knee OA than that of an unaffected limb or healthy controls.[21,22] Histologically, there is evidence of atrophy of fast twitch muscle fibres, muscular atrophy and both muscle fibre degeneration and regeneration.[23] Such morphologic changes may affect the mechanics of the muscle spindle, or changes in intrafusal muscle fibres of the spindle may mirror the changes to extrafusal muscle fibres. Other data highlight morphological and microstructural changes in muscle spindles of axial[24] and limb[25,26] muscles with age which have been speculated to affect their function.[27] Similar to general quadriceps muscle atrophy, such age-related change might be accelerated in knee OA. Whether muscle spindle function contributes to the proprioceptive deficit identified in knee OA has not been directly studied.

Muscle spindle function can be probed by investigation of the effect of muscle vibration. Vibration of ~60 Hz applied over either muscle or tendon excites muscle spindle afferents producing illusions of movement,[16,28–32] consistent with their role as muscle length/velocity transducers. When vibration is applied over axial and leg muscles in standing it generates illusions of falling, which in turn induce postural adjustments characterized by displacement of the centre of pressure (CoP). For example, vibration applied over the triceps surae muscles produces the illusion of muscle lengthening, and therefore apparent forward falling which provokes a reactive backwards lean and posterior shift of the CoP.[30,33,34]

Grant sponsor: National Health and Medical Research Council (NHMRC) of Australia; Grant number: ID631717; Grant sponsor: NHMRC Senior Principal Research Fellowship; Grant number: APP1002190.
Grant sponsor: Australian Research Council Future Fellowship.
*Correspondence to*: Paul Hodges (T: +61-7-3365-2008; F: +61-7-3365-2775; E-mail: p.hodges@uq.edu.au)

Perturbation of muscle spindles in quiet standing has been used to investigate both their role in proprioception and proprioceptive dysfunctions associated with musculoskeletal disorders.[29,31–33] Most studies of proprioceptive dysfunction in knee OA have involved conscious awareness of movement or position during passive or active movements of the limbs in sitting or standing.[8–15] Evaluation of the response to perturbation of sensory receptors in quiet standing enables assessment of proprioceptive mechanisms with minimal requirement for conscious control or response to the task. This type of assessment provides information of the same sensory system but reduces the potential impact of cognitive factors.

Quiet standing requires control of the body's centre of mass (CoM) relative to the base of support[35,36] and is under complex neuromotor control involving adaptive strategies to overcome perturbation of stable quiet standing that is signaled by the various sensory systems including the muscle spindles. Ankle and hip muscles are the most critical to detect and react to perturbation, but other leg, trunk and neck muscles contribute.[37,38] Although the contribution of the knee to quiet stance is less than the ankle and hip induced pain around the knee increases displacement of CoP in quiet stance.[39]

An important consideration for investigation of quiet standing is that deficits in proprioception related to knee OA might be compensated by other sensory information. Sensory information from multiple sources (vision, vestibular, somatosensory) is used to provide information on position and movement of the body and the environment[40,41] and this information is integrated as necessary to maintain postural control. When the availability or accuracy of a source of sensory input is compromised the nervous system can place greater reliance on other sources of information by a process termed "sensory reweighting."[40,41] Failure to appropriately reweight sensory information could compromise postural control.[40,41] Further, in the presence of loss/compromise of one sensory source (e.g., vestibular loss[40]) postural control may be compromised if dependence on that sensation is demanded by compromise to an additional source. For instance, impairment of quadriceps muscle spindle function in knee OA would compromise the accuracy or availability of sensory information from this source. This may not be problematic when this can be compensated by sensory information from other sources (e.g., vision and ankle muscles), but could underpin balance deficits if information from those sources was limited, requiring "reweighting" to knee muscle inputs. Such compromise to sensory reweighting (inability to reweight to reliance on sensory input from knee muscles) could explain the decreased ability of people with knee OA to adapt to changing sensory inputs from visual or somatosensory sources in quiet standing.[42]

This study aimed to test whether muscle spindle function of the quadriceps muscle (the muscle most affected in knee OA), and the triceps surae and tibialis anterior muscles (thought to be minimally affected in knee OA) is altered in people with knee OA. We hypothesized that vibration applied to the quadriceps muscle would induce greater displacement of the CoP in control participants than in those with knee OA (who we predicted would have less sensitive spindle afferents in this muscle). Further, we hypothesized that deficits in proprioceptive input from knee muscles would lead to greater displacement of CoP (greater perturbation) in response to vibration of triceps surae and tibialis anterior muscles secondary to compromised ability of the postural control system to reweight to sensory input from the knee muscles when vision is occluded and sensory information from ankle muscles is distorted.

## METHODS

### Participants

Thirty individuals with moderate/severe right knee OA (17 female, 66 (7) [mean(SD)] years) and 30 healthy asymptomatic controls (17 female, 65(8) years) participated. Age did not differ between groups (independent $t$-test; $p = 0.71$). Participants in the OA group had tibiofemoral joint OA in the right knee fulfilling the American College of Rheumatology (ACR) classification criteria[43] and an average knee pain $\geq$3 on an 11-point (0–10) numeric rating scale on most days of the month prior to enrolment (group mean (SD) −4.7(1.8)). OA participants were included if they had a Kellgren and Lawrence (KL) grade of 3 or 4 on weight-bearing X-ray (KL Grade 4 for $n = 21$).[44] KL grading/screening was performed by one of two researchers trained in the KL grading system and used radiographs taken within 12 months of the participant's enrolment in the study. Participants were included in the Control group if they had no knee pain or knee injury in the past 5 years. Volunteers were excluded if they had signs or symptoms of other pathology, including co-existing pathology that could hinder participation or performance (e.g., neurological conditions, lower limb surgery, systemic arthritis). The local Human Research Ethics Committee approved the study and all participants provided written informed consent.

### Ground Reaction Forces

Ground reaction forces were collected using an AMTI OR6-6-2000 strain gauge force plate (Advanced Mechanical Technologies, Inc., Watertown, MA) embedded in the laboratory floor. Force plate data were sampled at 120 Hz and 16-bit resolution using a Vicon motion analysis system (Vicon, Oxford, UK) and Nexus software.

### Procedure

Participants stood barefoot on a force plate with their feet a comfortable distance apart and their arms relaxed by their sides. Vision was occluded using a blindfold as vision is known to negate illusions of movement due to muscle spindle vibration.[30] Participants were asked to stand still but relaxed throughout the trials. Data were collected in three conditions in randomized order with vibration applied bilaterally over: (i) vastus medialis (approximately 15 cm proximal to the knee joint line), (ii) triceps surae (over Achilles tendon), or (iii) tibialis anterior (over the tibialis anterior muscle belly) muscles, sites previously shown to induce perturbation in

quiet standing, [31,32] as shown in Figure 1. Data were recorded for 45 s with three 15 s epochs: Baseline, Vibration, and Recovery. Vibration at 60 Hz was applied using custom-built devices that involved motor driven eccentric masses in a small plastic housing (plastic housing 94 mm × 44 mm × 32 mm, motor 36 mm × 17 mm × 15 mm), with an amplitude of displacement of approximately 0.5 mm. Each condition was repeated in two consecutive trials separated by a break of approximately 30 s.

### Data Analysis

CoP position was calculated with Nexus software using the ground reaction force data from the Vicon motion analysis system. Analysis of CoP was limited to the sagittal/anterior–posterior (A–P) direction as the vibration was applied to muscles that control motion in this direction and CoP displacements have been shown to be greatest in this direction.[31,45] Position of the CoP in the sagittal plane was averaged for each 1-s of the 45 s trial and expressed with respect to the average baseline CoP position in the sagittal direction averaged over the entire 15-s pre-vibration period using Matlab (The Mathworks, Inc., Nattick, MA). No digital filtering was applied prior to the 1-s averaging. Several features of the CoP response were identified from the data for each individual participant for descriptive purposes. These were: (i) peak CoP displacement during Vibration (largest displacement of the CoP from baseline average), and (ii) time to reach peak displacement during Vibration (1-s period in which the peak CoP displacement was identified).

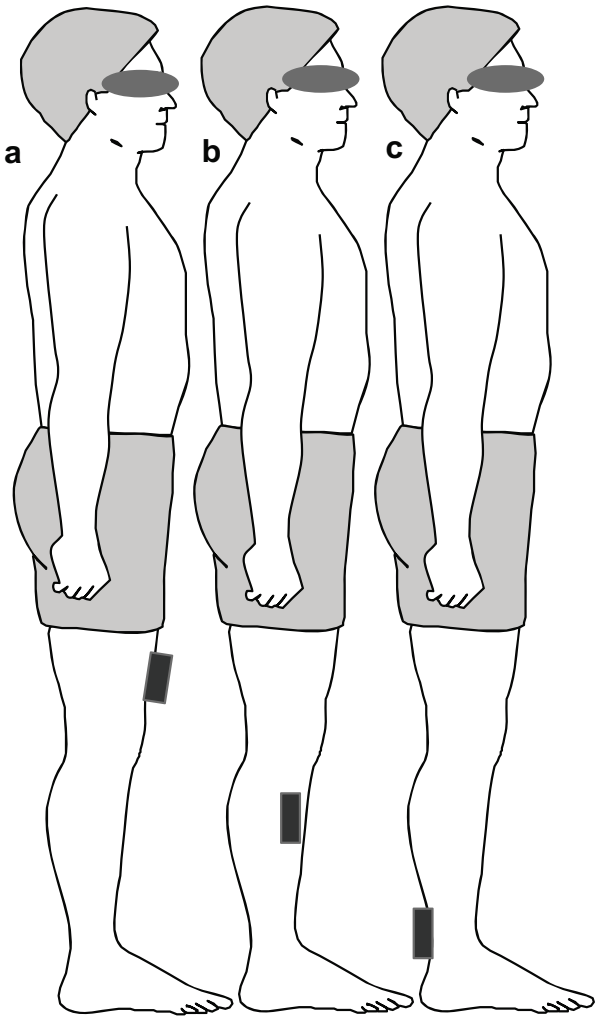


**Figure 1.** Experimental setup. Location for vibration applied to the: (a) quadriceps, (b) tibialis anterior and, (c) triceps surae. Testing of the three muscles was performed in randomized order.

### Statistical Analysis

Repeated measures analysis of variance (ANOVA) was used to compare CoP position between Epochs (Baseline vs. Vibration vs. Recovery; within subject factor), Time (fifteen consecutive 1-s measures during each epoch; within subject factor), Repetition (first vs. second; within subject factor), and Group (knee OA vs. Control; between-subjects factor). A separate ANOVA was used for each muscle. Where interactions were revealed post hoc analysis was conducted using Bonferroni adjustment or Bonferroni post hoc test.

The level of significance was set at $p \leq 0.05$. Statistical analyses were performed using IBM SPSS Statistics for Mac, Version 20 (IBM Corp., Armonk, NY) and STATISTICA (Statsoft, Inc., Tulsa, OK). Data are presented as mean (standard error) throughout the text and figures.

## RESULTS

Vibration over each muscle site (quadriceps, tibialis anterior and triceps surae) induced A–P CoP displacement (main effect—Epoch: all muscles $p < 0.01$) but the direction differed between muscles. CoP displaced posteriorly during vibration of the triceps surae, the position of CoP (mean (SEM)) relative to that at baseline and averaged across Groups, Repetitions, and Times was (24.3 (1.1) mm) and anteriorly during vibration of the quadriceps (0.8 (0.5) mm) and tibialis anterior (1.4 (0.9) mm) muscles (Figs. 2 and 3). Following removal of vibration (Recovery Epoch) CoP displaced in the opposite direction to Vibration for all muscles. Mean CoP position was more posterior during Recovery than Baseline and Vibration for quadriceps (3.6 (0.8) mm; post hoc—both $p < 0.001$) and tibialis anterior (2.7 (0.8) mm; post hoc—both $p \leq 0.01$) and more posterior during Vibration than Baseline and Recovery for triceps surae (8.2 (1.0) mm; post hoc—both $p < 0.001$) (Bonferroni adjustment). Mean CoP position did not differ between groups during any Epoch for trials with vibration to the quadriceps (main effect–Group: $p = 0.76$, interaction—Group × Epoch: $p = 0.69$), or tibialis anterior (main effect—Group: $p = 0.58$, interaction—Group × Epoch: $p = 0.78$).

The response to vibration over the triceps surae differed between the first and second repetition and this differed between groups (Group × Epoch × Repetition × Time: $p = 0.006$). The initial amplitude of perturbation in response to vibration was less for controls, reaching the peak displacement at a later time for the second Repetition for this group (Fig. 1c). Post-hoc analysis (Bonferroni Post Hoc test) revealed less difference (i.e., less adaptation) between repetitions of the trial for the OA group (OA—significant difference between first and second Repetition only from the third to fifth 1-s period during vibration [post hoc $p < 0.05$]; Control—significant difference between

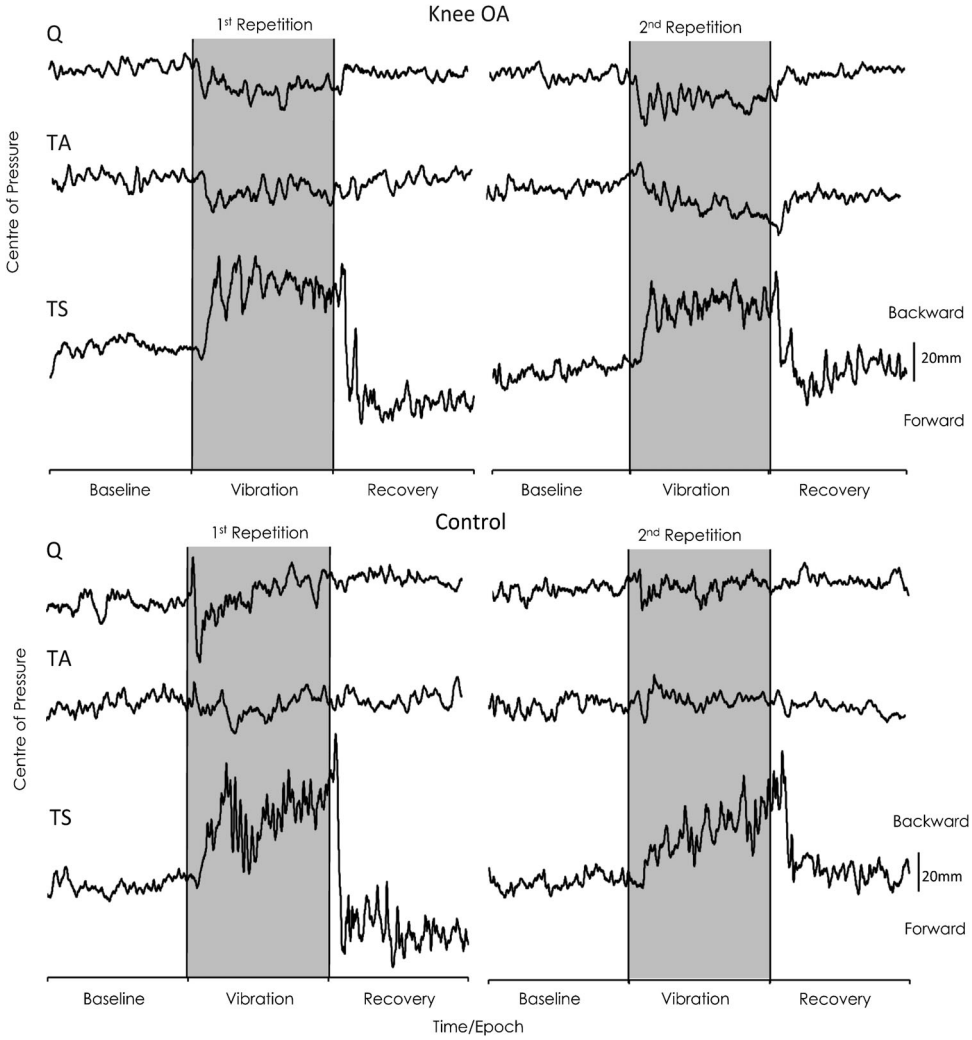


**Figure 2.** Raw data of COP A–P displacement for a representative participant in each group, for trials with vibration applied to the quadriceps (Q), tibialis anterior (TA), and triceps surae (TS). Note the greater attenuation of the perturbation due to vibration at the beginning and middle of the vibration Epoch in the 2nd Repetition for the Control participant than the OA participant.

Repetitions from the second to fourteenth 1-s period [post hoc $p < 0.05$]). During the second Repetition the OA participants were perturbed further than the controls in the second and 14th second during the vibration phase [post hoc $p < 0.05$]). Inspection of the data in Fig. 3 (area indicated by the "#") implies a difference in the rate of change of CoP towards the peak displacement between groups in the second Repetition. This was investigated in a secondary analysis of rate of change of position between groups, quantified as the slope of the regression line fitted to the CoP position versus time data (during the near-linear component of the CoP displacement between the 20th and 29th second). Comparison of these slopes indicated a faster rate of change in CoP position for OA participants (1.2 (0.2) $\text{mms}^{-1}$) than Controls (0.7 (0.2) $\text{mms}^{-1}$) ($t$-test—$p = 0.05$) in the second repetition.

Post hoc testing (Bonferroni Post Hoc tests) revealed CoP position differed between Groups and Repetitions during the Recovery epoch for the triceps surae muscle (between Group differences were identified between the 33rd and 34th second of the trial, $p < 0.05$, between Repetitions between the 33rd and 45th seconds of the trial, $p < 0.05$). Although the CoP tended to rebound anteriorly beyond the initial position after removal of the vibration in the first Repetition for both groups, this only persisted for the control group in the second Repetition. In the second Repetition CoP did not rebound beyond the initial CoP position for OA participants, consequently CoP position was less anterior during most 1-s periods during the second Repetition compared to the first (13 out of 15 Epochs, $p < 0.05$). Control participants reached a greater peak anterior displacement of CoP than OA participants in the second, or third and fourth seconds of the post-vibration epoch in the first or second Repetition, respectively.

## DISCUSSION

The results of this study did not support the hypothesis that vibration of the quadriceps muscle would induce smaller CoP displacement in people with OA than controls. Instead we found changes in the

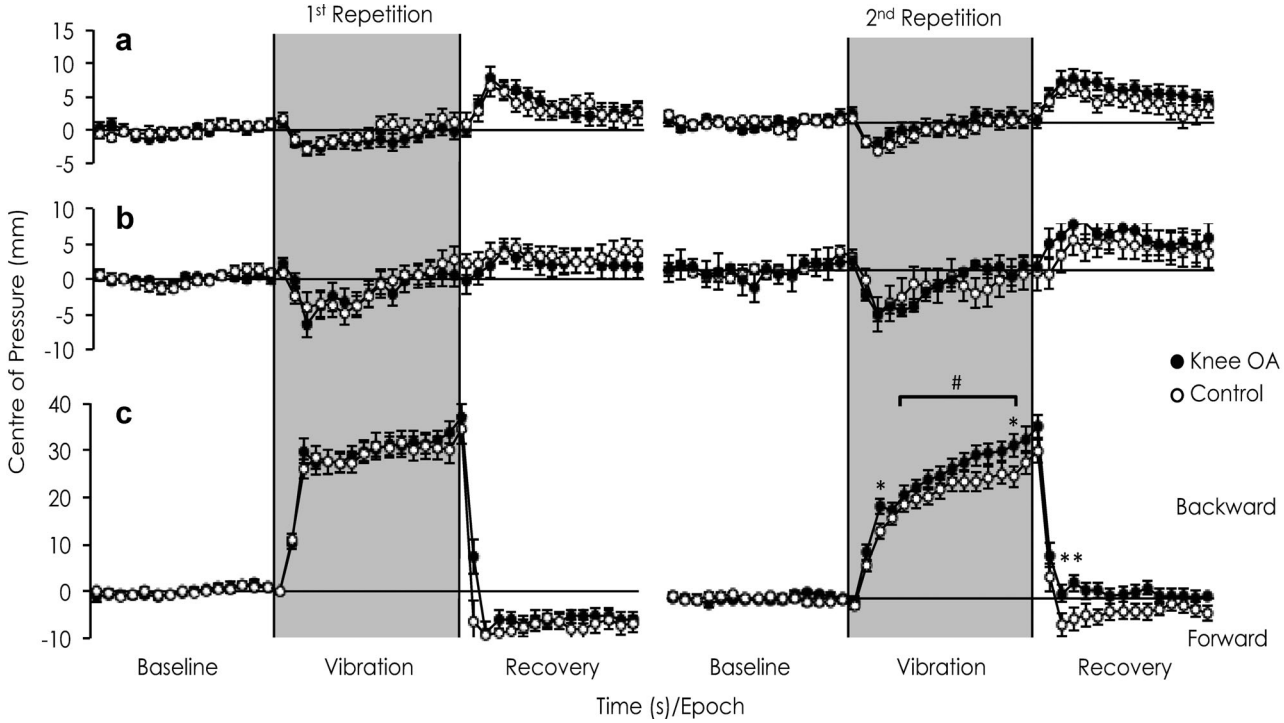


**Figure 3.** Group mean anteroposterior position of the CoP before (Baseline), during (Vibration), and after (Recovery) application of vibration to the quadriceps (a), tibialis anterior (b), and triceps surae (c) in knee OA and control groups. Data are shown separately for the first and second repetitions of the application of vibration. Bars denote standard error. CoP position was different between Vibration and Recovery for all three muscles. CoP position was also different between Baseline and Vibration for Triceps Surae with the same differences between Epochs for 2nd Repetition (not shown) as 1st Repetition. *Differences between groups for individual 1-s Time points, #significant difference in slopes between groups ($p < 0.054$).

response to vibration of the ankle plantarflexor muscles. Participants with OA had an exaggerated initial response to vibration of triceps surae and less adaptation to repeated exposure to vibration than Controls. An interpretation of these observations is that the OA participants are more dependent on information from the triceps surae muscles for postural control in quiet standing than controls, and information from the knee that could compensate was either not available or could not be used when erroneous information from the triceps surae muscle was provided by the vibration stimulus. That is, people with knee OA had compromised ability to reweight sensory information to other sources, which would include the quadriceps muscle in healthy individuals. Thus, although participants did not react differently to quadriceps vibration, when taken together the observations may reflect a deficit in quadriceps muscle spindle function.

### Postural Response to Quadriceps Muscle Vibration

We hypothesized that quadriceps muscle changes associated with knee OA[21–23] would lead to reduced responsiveness of the quadriceps muscle spindles in knee OA and subsequent reduction of CoP displacement in response to quadriceps vibration. Previous work has shown decreased balance perturbation from back muscle spindle stimulation and greater responsiveness to vibration applied to the triceps surae and tibialis anterior in low back pain, another condition associated with proprioceptive impairments including postural instability, and reduced lumbosacral proprioception.[33,46] The failure to observe similar changes for vibration over muscles at the site of pathology (i.e., quadriceps muscle) in the present study might be explained by the limited contribution of somatosensory information from the knee and quadriceps muscle to balance control in quiet stance. This would be consistent with the limited requirement for motion of the knee in quiet stance.[35] This would concur with the demonstration of minimal impact of knee immobilization on measures of postural control (including CoP displacements) in quiet standing.[38] The limited impact of perturbation of quadriceps input to balance control in either groups was perhaps not surprising in the presence of conflicting, undistorted somatosensory information from the triceps surae that likely indicated there was no perturbation to balance. Alternatively, the data could imply that there is no dysfunction of quadriceps muscle spindles or processing of muscle spindle information associated with severe knee OA. However, the response to triceps surae vibration questions this interpretation.

### Modified Response to Triceps Surae Muscle Spindle Stimulation in Knee OA

An unexpected finding of this study was the difference between the groups in response to triceps surae muscle spindle stimulation; the initial response in

participants with knee OA was more pronounced and the rate of change of CoP position was faster during the phase of near linear CoP displacement in the second repetition of vibration. Furthermore, there was greater attenuation of the response to triceps surae vibration in the second repetition by the Control group. Taken together, these observations suggest that control participants were more able to adapt to the artificially induced information from their ankle muscles. The likely explanation for this is that healthy participants were able to reweight the integration of sensory information. In this way distorted input from triceps surae could be compensated by other undistorted input from any of a number of sources.[40,41] Vision is not a candidate in this study as participants had vision occluded, but vestibular or somatosensory information from other segments/muscles, including the quadriceps could be involved. This reweighting of sensory information on the basis of incongruence of the artificial input due to vibration and input from other sensory sources (vestibular and somatosensory information from other muscles and body segments) is similar to that reported for other healthy groups in the presence of distortion or reduction of a source of sensory input.[33,47,48] Consistent with this hypothesis, it has also been shown that knee OA is associated with a decreased ability to adapt to changing sensory inputs from vision or surface conditions in quiet standing[42]; again suggesting a compromised ability to reweight between various sources of sensory input.

Although the present experiment does not permit interpretation of the mechanism for reduced adaptation and sensory reweighting in the knee OA group, several hypotheses can be proposed. First, although somatosensory information from the quadriceps usually contributes little to postural control (as evidenced by the relatively minor perturbation to CoP induced by vibration to this muscle), when somatosensory input from triceps surae is conflicted with other sensory sources, the somatosensory input from quadriceps may be required to be up-regulated to compensate. However, in the presence of deficits in knee proprioception in this group (including possible compromised muscle spindles), compensation may not be possible or erroneous somatosensory information may be provided from the knee. Second, the exaggerated and less adapted response to triceps surae vibration may be explained by altered responsiveness of spindles in this muscle. Although the mechanism for such a change is unclear, there may be changes to triceps surae structure and/or function associated with the knee OA disease process. Joint effusion/injury has been argued to reduce excitability of alpha motoneurons[49] and muscle spindle sensitivity[50] in muscles acting across the joint (i.e., arthrogenous muscle inhibition (AMI)).[51] This is thought to contribute to quadriceps weakness and impaired proprioception.[51] As the gastrocnemius muscle also crosses the knee joint, AMI may also impact motoneuron excitability (affecting the amplitude of gastrocnemius muscle response to recover balance) and muscle spindle sensitivity (reducing the response to Achilles tendon vibration) of that muscle in people with knee OA. However, this is unlikely to explain the present results as the response to Achilles tendon vibration was greater, not smaller, in people with knee OA than controls, and weakness of triceps surae is not a feature of knee OA.[52] Third, this difference in response could be explained by differences in central processing of sensory information. Previous work has shown sensory reorganization and processing issues in chronic musculoskeletal pain conditions.[33,53] It is unclear why this would involve the triceps surae and not the quadriceps muscle. Two possible explanations for the involvement of the triceps surae are: (1) that the gastrocnemius muscle crosses the knee joint and thus contributes to knee function and, (2) differences in muscle activation patterns of the gastrocnemius muscles have been reported in people with knee OA.[54]

### Limitations

A number of methodological limitations require consideration. First, all participants in the OA group had moderate to severe knee OA (KL grade 3 or 4) with pain ratings of $\geq$3/10, and as such the findings cannot be generalized to knee OA populations with less severe OA. Second, this study does not allow direct inference as to whether the changes in the proprioceptive system are a result of the knee OA disease process or how the muscle spindles may be involved. Last, although we observed changes in quiet standing, further work is required to determine whether these changes affect more dynamic situations.

## CONCLUSION

The present findings add to the understanding of proprioceptive impairments in OA and provide insight into broader application of deficit in this system. Although the study did not find a difference between groups when quadriceps muscle spindle input was manipulated, the response to calf muscle vibration was modified. Our interpretation is that quadriceps proprioceptive information provides little contribution to control of quiet standing when information is available from other sources (and therefore, perturbation to quadriceps input has little impact on balance). However, when the major sensory contribution from triceps surae is challenged, people with knee OA have less potential to detect or compensate for this (potentially as a result of compromised function of quadriceps muscle spindles) and as a result are distorted to a greater effect by challenges to triceps surae sensory input, and adapt to this perturbation less efficiently. This novel interpretation has potentially important implications for more demanding functions and requires further investigation.

## ACKNOWLEDGMENTS

The study was funded by a Program Grant (ID631717) from the National Health and Medical Research Council (NHMRC)

of Australia. Paul Hodges is supported by an NHMRC Senior Principal Research Fellowship (APP1002190). Kim Bennell is partly supported by an Australian Research Council Future Fellowship.

## REFERENCES


1. Hunter DJ, McDougall JJ, Keefe FJ. 2008. The symptoms of osteoarthritis and the genesis of pain. Rheum Dis Clin North Am 34:623–643.
2. Felson DT, Gross KD, Nevitt MC, et al. 2009. The effects of impaired joint position sense on the development and progression of pain and structural damage in knee osteoarthritis. Arthritis Care Res 61:1070–1076.
3. Van Der Esch M, Steultjens M, Harlaar J, et al. 2007. Joint proprioception, muscle strength, and functional ability in patients with osteoarthritis of the knee. Arthritis Care Res 57:787–793.
4. Sharma L, Cahue S, Song J, et al. 2003. Physical functioning over three years in knee osteoarthritis: role of psychosocial, local mechanical, and neuromuscular factors. Arthritis Rheum 48:3359–3370.
5. Smith TO, King JJ, Hing CB. 2012. The effectiveness of proprioceptive-based exercise for osteoarthritis of the knee: a systematic review and meta-analysis. Rheumatol Int 32:3339–3351.
6. Marks R. 1994. An investigation of the influence of age, clinical status, pain and position sense on stair walking in women with osteoarthrosis. Int J Rehabil Res 17:151–158.
7. Knoop J, Steultjens MPM, van der Leeden M, et al. 2011. Proprioception in knee osteoarthritis: a narrative review. Osteoarthritis Cartilage 19:381–388.
8. Barrack RL, Skinner HB, Cook SD, et al. 1983. Effect of articular disease and total knee arthroplasty on knee joint-position sense. J Neurophysiol 50:684–687.
9. Barrett DS, Gobb AG, Bentley G. 1991. Joint proprioception in normal, osteoarthritic and replaced knees. J Bone Joint Surg Br 73:53–56.
10. Marks R, Quinney HA, Wessel J. 1993. Proprioceptive sensibility in women with normal and osteoarthritic knee joints. Clin Rheumatol 12:170–175.
11. Sell S, Zacher J, Lack S. 1993. Proprioception decline in the osteoarthritic knee. Z Rheumatol 52:150–155.
12. Hurley MV, Scott DL, Rees J, et al. 1997. Sensorimotor changes and functional performance in patients with knee osteoarthritis. Ann Rheum Dis 56:641–648.
13. Hassan BS, Mockett S, Doherty M. 2001. Static postural sway, proprioception, and maximal voluntary quadriceps contraction in patients with knee osteoarthritis and normal control subjects. Ann Rheum Dis 60:612–618.
14. Sharma L, Pai YIC, Holtkamp K, et al. 1997. Is knee joint proprioception worse in the arthritic knee versus the unaffected knee in unilateral knee osteoarthritis? Arthritis Rheum 40:1518–1525.
15. Lund H, Juul-Kristensen B, Hansen K, et al. 2008. Movement detection impaired in patients with knee osteoarthritis compared to healthy controls: a cross-sectional case-control study. J Musculoskelet Neuronal Interact 8:391–400.
16. Cordo P, Gurfinkel VS, Bevan L, et al. 1995. Proprioceptive consequences of tendon vibration during movement. J Neurophysiol 74:1675–1688.
17. Goodwin GM, McCloskey DI, Matthews PB. 1972. The contribution of muscle afferents to kinaesthesia shown by vibration induced illusions of movement and by the effects of paralysing joint afferents. Brain 95:705–748.
18. Inglis JT, Frank JS, Inglis B. 1991. The effect of muscle vibration on human position sense during movements controlled by lengthening muscle contraction. Exp Brain Res 84:631–634.
19. Roll JP, Vedel JP. 1982. Kinaesthetic role of muscle afferents in man, studied by tendon vibration and microneurography. Exp Brain Res 47:177–190.
20. Proske U, Gandevia SC. 2009. The kinaesthetic senses. J Physiol (Lond) 587:4139–4146.
21. Ikeda S, Tsumura H, Torisu T. 2005. Age-related quadriceps-dominant muscle atrophy and incident radiographic knee osteoarthritis. J Orthop Sci 10:121–126.
22. Petterson SC, Barrance P, Buchanan T, et al. 2008. Mechanisms underlying quadriceps weakness in knee osteoarthritis. Med Sci Sports Exerc 40:422–427.
23. Fink B, Egl M, Singer J, et al. 2007. Morphologic changes in the vastus medialis muscle in patients with osteoarthritis of the knee. Arthritis Rheum 56:3626–3633.
24. Boyd-Clark LC, Briggs CA, Galea MP. 2002. Muscle spindle distribution, morphology, and density in longus colli and multifidus muscles of the cervical spine. Spine 27:694–701.
25. Swash M, Fox KP. 1972. The effect of age on human skeletal muscle studies of the morphology and innervation of muscle spindles. J Neurol Sci 16:417–432.
26. Liu JX, Eriksson PO, Thornell LE, et al. 2005. Fiber content and myosin heavy chain composition of muscle spindles in aged human biceps brachii. J Histochem Cytochem 53:445–454.
27. Shaffer SW, Harrison AL. 2007. Aging of the somatosensory system: a translational perspective. Phys Ther 87:193–207.
28. Goodwin GM, McCloskey DI, Matthews PB. 1972. A systematic distortion of position sense produced by muscle fibration. J Physiol (Lond) 221:8P–9P.
29. Lackner JR, Levine MS. 1979. Changes in apparent body orientation and sensory localization induced by vibration of postural muscles: vibratory myesthetic illusions. Aviat Space Environ Med 50:346–354.
30. Eklund G. 1972. General features of vibration induced effects on balance. Ups J Med Sci 77:112–124.
31. Ivanenko YP, Grasso R, Lacquaniti F. 2000. Influence of leg muscle vibration on human walking. J Neurophysiol 84:1737–1747.
32. Courtine G, De Nunzio AM, Schmid M, et al. 2006. Stance- and locomotion-dependent processing of vibration-induced proprioceptive inflow from multiple muscles in humans. J Neurophysiol 97:772–779.
33. Brumagne S, Cordo P, Verschueren S. 2004. Proprioceptive weighting changes in persons with low back pain and elderly persons during upright standing. Neurosci Lett 366:63–66.
34. Ivanenko YP, Solopova IA, Levik YS. 2000. The direction of postural instability affects postural reactions to ankle muscle vibration in humans. Neurosci Lett 292:103–106.
35. Winter DA. 1995. Human balance and posture control during standing and walking. Gait Posture 3:193–214.
36. Maki BE, McIlroy WE, Fernie GR. 2003. Change-in-support reactions for balance recovery. IEEE Eng Med Biol Mag 22:20–26.
37. Gage WH, Winter DA, Frank JS, et al. 2004. Kinematic and kinetic validity of the inverted pendulum model in quiet standing. Gait Posture 19:124–132.
38. de Freitas PB, Freitas SM, Duarte M, et al. 2009. Effects of joint immobilization on standing balance. Hum Mov Sci 28:515–528.
39. Hirata RP, Arendt-Nielsen L, Shiozawa S, et al. 2011. Experimental knee pain impairs postural stability during quiet stance but not after perturbations. Eur J Appl Physiol 112:1–11.
40. Horak FB, Shupert CL, Mirka A. 1989. Components of postural dyscontrol in the elderly: a review. Neurobiol Aging 10:727–738.
41. Carver S, Kiemel T, Jeka JJ. 2006. Modeling the dynamics of sensory reweighting. Biol Cybern 95:123–134.

42. Hirata RP, Jorgensen TS, Rosager S, et al. 2013. Altered visual and feet proprioceptive feedbacks during quiet standing increase postural sway in patients with severe knee osteoarthritis. PLoS ONE 8.
43. Altman R, Asch E, Bloch D. 1986. Development of criteria for the classification and reporting of osteoarthritis. Classification of osteoarthritis of the knee. Arthritis Rheum 29:1039–1052.
44. Kellgren JH, Lawrence JS. 1957. Radiological assessment of osteo-arthrosis. Ann Rheum Dis 16:494–502.
45. Courtine G, Pozzo T, Lucas B, et al. 2001. Continuous, bilateral Achilles' tendon vibration is not detrimental to human walk. Brain Res Bull 55:107–115.
46. Brumagne S, Cordo P, Lysens R, et al. 2000. The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain. Spine 25:989–994.
47. Woollacott MH, Shumway-Cook A, Nashner LM. 1986. Aging and posture control: changes in sensory organization and muscular coordination. Int J Aging Hum Dev 23:97–114.
48. Bugnariu N, Fung J. 2007. Aging and selective sensorimotor strategies in the regulation of upright balance. J NeuroEng Rehabil 4.
49. Spencer JD, Hayes KC, Alexander IJ. 1984. Knee joint effusion and quadriceps reflex inhibition in man. Arch Phys Med Rehabil 65:171–177.
50. Freeman MA, Wyke B. 1967. Articular reflexes at the ankle joint: an electromyographic study of normal and abnormal influences of ankle-joint mechanoreceptors upon reflex activity in the leg muscles. Br J Surg 54:990–1001.
51. Hurley MV, Newham DJ. 1993. The influence of arthrogenous muscle inhibition on quadriceps rehabilitation of patients with early, unilateral osteoarthritic knees. Br J Rheumatol 32:127–131.
52. Rutherford DJ, Hubley-Kozey CL, Stanish WD. 2011. Maximal voluntary isometric contraction exercises: a methodological investigation in moderate knee osteoarthritis. J Electromyogr Kinesiol 21:154–160.
53. Simoneau M, Mercier P, Blouin J, et al. 2006. Altered sensory-weighting mechanisms is observed in adolescents with idiopathic scoliosis. BMC Neurosci 7.
54. Childs JD, Sparto PJ, Fitzgerald GK, et al. 2004. Alterations in lower extremity movement and muscle activation patterns in individuals with knee osteoarthritis. Clin Biomech 19: 44–49.